Research Article

Arm crank ergometry improves cardiovascular disease risk factors and community mobility independent of body composition in high motor complete spinal cord injury

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Objective: Evaluate the effect of aerobic exercise using arm crank ergometry (ACE) in high motor complete (ISNCSCI A/B) spinal cord injury (SCI) as primarily related to cardiovascular disease (CVD) risk factors and functional mobility and secondarily to body composition and metabolic profiles.
Design: Longitudinal interventional study at an academic medical center.
Methods: Ten previously untrained participants (M8/F2, Age 36.7 y ± 10.1, BMI 24.5 ± 6.0) with high motor complete SCI (C7-T5) underwent ACE exercise training 30 minutes/day × 3 days/week for 10 weeks at 70% VO_{2Peak}.
Outcome Measures: Primary outcome measures were pre- and post-intervention changes in markers of cardiovascular fitness (graded exercise testing (GXT): VO_2, VO_{2Peak}, respiratory quotient [RQ], GXT time, peak power, and energy expenditure [EE]) and community mobility (time to traverse a 100ft-5° ramp, and 12-minute WC propulsion test). Secondary outcome measures were changes in body composition and metabolic profiles (fasting and area under the curve for glucose and insulin, homeostasis model assessment [HOMA] for %β-cell activity [%β], %insulin sensitivity [%S], and insulin resistance [IR], and Matsuda Index [$ISI_{Matsuda}$]).
Results: Resting VO_2, relative VO_{2Peak}, absolute VO_{2Peak}, peak power, RQ, 12-minute WC propulsion, fasting insulin, fasting G:I ratio, HOMA-%S, and HOMA-IR all significantly improved following intervention ($P < 0.05$). There were no changes in body composition ($P > 0.05$).
Conclusions: Ten weeks of ACE at 70% VO_{2Peak} in high motor complete SCI improves aerobic capacity, community mobility, and metabolic profiles independent of changes in body composition.

Introduction

Respiratory and renal conditions have historically been the most prevalent cause of morbidity and mortality in the spinal cord injury (SCI) population.[1] However, with improvements in medicine and our understanding in secondary complications that result from SCI, life expectancy has been on the rise and age-related comorbidities have become a greater determinant of survival. In fact, recent studies suggest that cardiovascular disease (CVD) is at greater prevalence in chronic SCI than the able-bodied (AB) population and is a leading cause of mortality in this population.[1–3]

Obesity is a well-recognized CVD risk factor and is more likely to occur in SCI because of the relative loss of metabolically active lean body mass (LBM), subsequent increase in body fat mass (FM), as well as blunting of the sympathetic nervous system.[4,5] These changes lead to greater percent fat mass (%FM) in sedentary SCI men and women when compared to physically active age and gender matched individuals with SCI, as well as AB

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controls.[2,6–9] Excess body fat has been shown to mediate metabolic syndrome via abnormal lipid and glucose profiles, cardiovascular inflammation, insulin resistance, hypertension, and thromboemboli.[9–12] Metabolic syndrome is widespread in the SCI population.[13–16] These risk factors have become targets of therapeutic interventions, such as exercise.[17]

The American College of Sports Medicine (ACSM) recommends 150 minutes of exercise per week to improve CVD risk factors.[18] SCI Action Canada recommends 40 minutes/week of moderate-to-vigorous aerobic physical activity, while the American Congress of Rehabilitation Medicine (ACRM) recommends ≥ 40–60 minutes/week of moderate-to-vigorous aerobic activity.[19,20] Both organizations call for more comprehensive exercise research in the SCI population.

Exercise limitations within the SCI population are numerous,[21] but an inexpensive and widely used exercise modality for this population is arm crank ergometry (ACE). A quantifiable measure of cardiovascular health and maximal cardiorespiratory work is peak oxygen consumption (VO_{2Peak}) which has previously been demonstrated as markedly reduced in SCI compared to the AB population.[22] VO_{2Peak} declines with age due to changes in body composition and cardiovascular health in the AB population.[23]

Only 25% of relatively young patients with SCI demonstrate aerobic capacity that is sufficient to meet the demands of independent mobility and living (> 15 ml/kg/min).[24] Moreover, in SCI above T6, VO_{2Peak} is inversely related to the level of injury (LOI) as a result of the limited volume of working abdominal musculature, ventilatory musculature, and circulatory dyskinesis.[25–27] Previous research has demonstrated that exercise in the SCI population can improve aerobic capacity.[25,27–31]

ACE has occasionally shown the ability to augment some measures of metabolic profiles,[32–35] however, these findings are inconsistent.[33,36,37] Upper body exercise appears to improve hepatic insulin sensitivity but the effect on peripheral insulin sensitivity is poorly understood.[38–40] The intensity, frequency, and duration of ACE needed to alter metabolic profiles is in need of further research.

Previous research on exercise in the SCI population has often failed to adequately control for completeness and level of injury. To our knowledge, no previous investigations have studied a range of CVD risk factors (GXT, two measures of community mobility, total and regional body composition, lipid profiles, and OGTT with measures of hepatic and peripheral insulin sensitivities) this extensively in high-motor complete injuries. In this study, we aimed to (1) evaluate the effects of ACE in high motor complete SCI as related to markers of aerobic fitness and community mobility and (2) examine effects of such exercise intervention on body composition and metabolic profiles. We hypothesized that following the ACE exercise intervention, markers of aerobic fitness, community mobility, body composition, and metabolic profiles would significantly improve.

Methods

Study design

Participants were recruited from the host institution to undergo aerobic exercise training and evaluation of CVD risk factors, which included aerobic capacity, community mobility, body composition, and serum metabolic profile markers for a longitudinal interventional study. All participants underwent complete physical examination by a physiatrist and 12-lead electrocardiogram prior to participation. Participants who met the inclusion criteria were defined as men and women, 18–55 years old with a C7-T4 motor complete SCI (ISNCSCI A or B) for greater than 6 months. Exclusion criteria included those individuals in an exercise program within the past 3 months; had known CVD, diabetes mellitus (type one or type two), hypothyroidism, and/or renal disease; uncontrolled autonomic dysreflexia; recent venous thromboembolism; pressure injury > grade II; or heterotopic ossification involving the upper extremities. This study was approved by the Intuitional Review Board at the host institution and all participants completed informed consent prior to the start of the study.

Exercise intervention

Qualified participants underwent ACE exercise training with a Monark Rehab Trainer 881E ACE (Patterson Medical, Warrenville,IL) for 30 minutes/day×3 days/week for 10 weeks at 70% VO_{2Peak}. 90-minutes per week was chosen to more closely mimic the ACSM exercise guidelines for AB individuals while taking into account the significant sympathetic and cardiopulmonary disadvantages.[18] In AB individuals, HR is a reliable indicator of oxygen uptake, however, in SCI individuals, this relationship does not hold true secondary to sympathetic blunting.[22] Training intensity was assigned in the current study on the basis of the peak power output and rate of perceived exertion (RPE) at 70% VO_{2peak}. VO_{2Peak} & peak power were re-assessed at the end of week 5 to allow any necessary adjustments in absolute exercise intensity to maintain relative intensity at the appropriate level. Exercise sessions occurred at the exercise physiology laboratory at the host institution. Each exercise training session had a 5-minute warm-up consisting of 5 watts ACE performed at 50 RPM and

a similar 5-minute cool-down period. Participants initially began exercising for shorter bouts with brief rest periods, allowing for the accumulation of total duration of 30 minutes of exercise. During the first week exercise sessions consisted of three 10-minute exercise bouts, while in the second week, exercise sessions consisted of two 15-minute bouts with a 5-minute rest period. From weeks 4–10 participants trained 30 minutes consecutively without rest at their designated intensity level. HR, BP, and RPE were monitored in each patient throughout every session. All subjects met physiological criteria for VO_{2Peak} during Pre- and Post-testing, including Peak HR with drop in SBP, Respiratory Exchange Ratio > 1.1 and Borg Rate of Perceived Exertion (RPE) > 19. Transportation was arranged for subjects. It was predetermined that subjects would be removed from participation if they missed more than six of the thirty sessions due to the aims of the current study.

Graded exercise testing

Participants underwent a maximal graded exercise test (GXT) with a Physio-Dyne Max-1® (AEI Technologies, Naperville, IL) metabolic measurement system. To determine resting oxygen consumption (VO_2), resting respiratory quotient (RQ; defined as the ratio of carbon dioxide production to oxygen consumption), and energy expenditure (EE) subjects were tested in a supine position and abided by the best practice guidelines for measurement of resting metabolic rates.[41] $VO_{2Peak,}$ GXT time, and peak power (PO_{peak}) were recorded with a standard ACE protocol one week before and within 4–7 days after the training intervention. Blood pressure was monitored throughout and heart rate was monitored via a lead II electrocardiogram (Quinton Q710 ECG system, Milwaukee, WI). Initial resistance of 5 watts was applied with metronome guided crank rate of 50 RPM. A warm-up stage of 2 minutes was used at this work rate, with subsequent 60-second stages of 5-watt increments employed until exhaustion. Respiratory exchange ratio, heart rate (HR), blood pressure (BP), and RPE was monitored during GXT. All participants were tested in their own stabilized wheelchair with appropriate seating, truncal stability, leg wraps, abdominal binder, and protective hand mitts that were secured to the ACE pedals.

Community mobility

Community mobility was assessed before and after the 10-week intervention with a 12-minute propelled distance performed on a 290-meter rubberized, level, indoor track as described and validated by Franklin *et al.*,[42] as well as timed ascent up a 100-foot 5° ramp. Testing was performed in a lightweight wheelchair (Quickie, Southwest Medical, Phoenix, Arizona) adjusted to the participants' height, arm length, and girth. All tests were timed and closely supervised by an exercise physiologist.

Body composition analysis and metabolic profiles

Body composition analysis was performed by measurement of total and regional values for the following variables: bone mineral content (BMC), bone mineral density (BMD), percent fat mass (%FM), fat mass (FM), and lean body mass (LBM) as determined by Dual Energy X-ray Absorptiometry (DXA; Lunar DPX-L, Lunar Corporation, Madison, Wisconsin) before and after the 10-week intervention. Every effort was taken to mimic the original participants' position on the scanner at both evaluation time points. Scans were performed after lying flat for at least 20 minutes to minimize fluid shift. All scans were performed and analyzed by a certified DXA operator using Lunar software.

After a 12-hour fast, an indwelling Teflon catheter (DuPont, Wilmington, Delaware) was placed in an antecubital vein of one arm to collect 4-ml blood sample that was used to collect total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), and triglycerides (TG).

A standard 75-g oral glucose tolerance test (OGTT) was administered between the hours 0700 and 1000 according to previously published methods.[43] Briefly, 3 mL blood samples were taken at rest and at 0, 30, 60, 90, 120, and 180 minutes after the ingestion of 100 g glucose in a 10-ounce solution to evaluate the responses of glucose, plasma insulin, and the glucose:insulin (G:I) ratio. All blood collected during the study was immediately placed on ice, transferred to a chemistry pathology laboratory, centrifuged, aliquoted, and analyzed in accordance with CDC and standard practice guidelines.

The serum concentrations of glucose, triglycerides, cholesterol, LDL-C, and HDL-C were determined using colorimetric assays at the host institution (General Clinical Research Center [GCRC] Core Laboratory, Lexington, KY), while plasma insulin concentration was measured using a radioimmunoassay kit (GCRC core laboratory, Lexington, KY). The glucose and insulin area under the curve (AUC) was computed via the trapezoidal rule.[43]

The Homeostatic Model Assessment (HOMA) of insulin resistance (IR), percent β-Cell function (%β), percent fasting insulin sensitivity (%S), and the Matsuda Insulin Sensitivity ($ISI_{Matsuda}$) were

calculated.[44,45] All carbohydrate and lipid measurements were performed one week before and 4-to-7 days after the 10-week intervention.

Statistical analysis

Normality was assessed with Shapiro-Wilks test. Wilcoxon signed rank tests were used to evaluate the effectiveness of exercise on GXT, body composition, community mobility, and metabolic biomarkers before and after the 10-week intervention. Level of significance was set at $P < 0.05$ and all analyses were performed with IBM SPSS Statistics 24 (PASW, SPSS Inc., IBM, Armonk, New York).

Results

Participant demographics

Ten patients (M8/F2; Age: 36.7 ± 12.5 years; BMI: 24.5 ± 6.0 kg/m^2) were included in the study. Thirty percent of the patients were classified as cervical injuries, while 70% were classified as high thoracic injuries. Eight patients were classified as ISNCSCI A and two were ISNCSCI B. Four participants dropped out of the study due to non-compliance. Specifically, these subjects had issues associated with work, family and community responsibilities, and social activities that took precedent over attending exercise sessions. Table 1 presents participant demographic data.

Graded exercise test

Post-intervention resting VO_2 (172.5 ± 50.0 vs. 195.3 ± 44.6 mL/min, $P = 0.046$), resting respiratory quotient (0.96 ± 0.15 vs. 0.77 ± 0.02, $P = 0.028$), absolute VO_{2peak} (784.2 ± 279.6 vs. 918.5 ± 310.0 mL/min, $P = 0.028$), relative VO_{2peak} (10.8 ± 3.6 vs. 12.8 ± 4.0 mL/kg/min, $P = 0.027$), and peak power (40 ± 16 vs. 54 ± 17 W, $P = 0.026$) significantly improved when compared to their baseline data. All other parameters did not change and are shown in Table 2.

Community mobility

The 12-minute wheelchair propulsion (2062 ± 1167 vs. 2398 ± 1260 feet, $P = 0.028$; Table 3) significantly increased following the exercise intervention. There was no significant difference between pre and post-intervention measurement of time to traverse a 100-foot 5° ramp (18.8 ± 7.8 vs. 18.2 ± 10.3 sec, $P = 0.463$; Table 3).

Body composition and metabolic profiles

There was no significant difference in body composition following the exercise intervention ($P > 0.05$; Table 4.) Post-intervention fasting insulin (12.23 ± 5.58 vs. 7.65 ± 2.34 μU/ml, $P = 0.028$), fasting G:I ratio (9.77 ± 4.49 vs. 13.69 ± 3.29, $P = 0.028$), HOMA-%S (73.3 ± 31.6 vs. 105.6 ± 27.1; $P = 0.046$) and HOMA-IR (1.6

Table 2. Graded exercise testing before and after intervention.

	Pre-intervention	Post-intervention	P-value
Resting VO_2 (ml/min)	172.5 ± 50.0	195.3 ± 44.6	0.046
VO_{2peak} (ml/min)[a]	784.2 ± 279.6	918.5 ± 310.0	0.028
VO_{2peak} (ml/kg/min)[b]	10.8 ± 3.6	12.8 ± 4.0	0.027
Peak Power (W)	40 ± 16	54 ± 17	0.026
GXT Time (min)[c]	23.2 ± 7.8	24.0 ± 4.7	0.920
Peak Heart Rate (BPM)	147.8 ± 23.2	151.2 ± 24.2	0.750
Resting Respiratory Quotient	0.96 ± 0.15	0.77 ± 0.02	0.028
Energy Expenditure (kcal/day)	1206.67 ± 336.19	1310.00 ± 301.13	0.075

[a]Absolute peak oxygen consumption; [b]relative peak oxygen consumption; [c]graded exercise test.

Table 1. Participant demographics.

Age	Sex	LOI	ISNCSCI	TSI (years)	Height (cm)	Weight (kg)		BMI (kg/m^2)	
						Pre	Post	Pre	Post
30	M	T2	A	30	168.0	100.5	NF	35.6	NF
52	F	T4	A	34	155.9	64.0	61.00	26.6	25.4
51	M	T1	A	32	188.0	66.0	68.30	18.7	19.3
22	M	T4	A	1	175.0	68.5	NF	22.3	NF
38	M	C7	A	0.6	185.0	109.5	112.50	32.0	32.7
26	M	T5	A	1	180.0	63.4	60.90	19.6	18.8
23	F	C7	B	5	168.0	50.0	50.00	17.7	17.7
36	M	C7	B	4	178.0	90.0	91.00	28.4	28.7
55	M	T2	A	15	180.3	67.3	NF	20.7	NF
26	M	T4	A	1.5	201.0	95.3	NF	23.6	NF
Mean									
36.7	NA	NA	NA	12.4	178.1	77.5	74.0	24.5	23.8

LOI, level of injury; ISNCSCI, international standard of neurological classification for spinal cord injury; TSI, time since injury; NF, no follow-up.

Table 3. Community mobility before and after intervention.

	n	Pre-intervention	Post-intervention	P-value
12-minute Propulsion (feet)	6	2062 ± 1167	2398 ± 1260	0.028
100 feet-5° Incline (seconds)	6	18.8 ± 7.8	18.2 ± 10.3	0.463

WC, wheelchair.

Table 4. Body composition.

		Pre-intervention	Post-intervention	P-value
Arms	FM (g)	1509 ± 1026	1520 ± 756	0.600
	LBM (kg)	4.12 ± 1.50	4.33 ± 1.64	0.917
	%FM	26.85 ± 15.54	26.37 ± 14.00	0.600
	BMC (g)	300 ± 115	345 ± 126	0.249
	BMD (g/cm^2)	1.02 ± 0.16	1.04 ± 0.17	0.345
Legs	FM (g)	8336 ± 3696	8273 ± 3916	0.345
	LBM (kg)	12.35 ± 33.51	12.47 ± 38.74	0.917
	%FM	39.45 ± 9.36	38.93 ± 8.44	0.225
	BMC (g)	717 ± 467	698 ± 393	0.600
	BMD (g/cm^2)	0.96 ± 0.34	0.96 ± 0.32	0.753
Trunk	FM (g)	13, 671 ± 7521	13, 574 ± 7108	0.917
	LBM (kg)	25.20 ± 63.43	25.18 ± 67.91	0.600
	%FM	33.23 ± 10.68	31.95 ± 8.99	0.753
	BMC (g)	1063 ± 331	1064 ± 324	0.753
	BMD (g/cm^2)	0.96 ± 0.09	0.96 ± 0.09	1.000
Total Body	FM (g)	25, 059 ± 11, 891	24, 810 ± 11, 826	0.753
	LBM (kg)	44.31 ± 10.26	44.83 ± 11.41	0.753
	%FM	34.91 ± 34.91	34.46 ± 34.46	0.345
	BMC (g)	2635 ± 869	2661 ± 879	0.463
	BMD (g/cm^2)	1.13 ± 0.15	1.12 ± 0.15	0.752

%FM, % fat mass; BMC, bone mineral content; BMD, bone mineral density; FM, fat mass; LBM, lean body mass.

± 0.7 vs. 1.0 ± 0.3; P = 0.046) significantly improved from baseline, while HOMA-%β, Glucose AUC, Insulin AUC, and $ISI_{Matsuda}$ did not significantly differ following the exercise intervention (P > 0.05). TG profiles improved, but did not reach significance following the intervention (P > 0.05). Metabolic profile measurements are presented in Table 5.

Discussion

This study focused on evaluating the effects of ten weeks of ACE on cardiovascular disease risk factors and community mobility. The main findings from this study suggest ten weeks of ACE significantly improve markers of aerobic fitness, 12-minute wheelchair propulsion, and some metabolic markers of cardiovascular

Table 5. Metabolic profiles before and after intervention.

	Pre-intervention	Post-intervention	P-value
Total cholesterol (mg/dl)	174.00 ± 22.69	175.50 ± 16.50	0.750
HDL-C (mg/dl)	36.33 ± 6.31	34.83 ± 7.31	0.066
% HDL-C	21.33 ± 4.03	19.83 ± 4.17	0.074
LDL-C (mg/dl)	104.83 ± 14.93	116.83 ± 17.98	0.116
TG (mg/dl)	164.50 ± 132.05	120.00 ± 57.85	0.600
OGTT			
Fasting glucose (mg/dl)	99.83 ± 14.80	99.00 ± 10.43	0.917
Fasting Insulin (μU/ml)	12.23 ± 5.58	7.65 ± 2.34	0.028
Fasting G:I	9.77 ± 4.49	13.69 ± 3.29	0.028
Glucose AUC	914.67 ± 236.60	968.58 ± 236.60	0.249
Insulin AUC	440.88 ± 212.88	464.76 ± 152.95	0.917
HOMA and $ISI_{Matsuda}$			
HOMA-%β	111.4 ± 48.7	82.4 ± 19.1	0.116
HOMA-%S	73.3 ± 31.6	105.6 ± 27.1	0.046
HOMA-IR	1.6 ± 0.7	1.0 ± 0.3	0.046
$ISI_{Matsuda}$	3.4 ± 1.6	3.6 ± 0.8	0.345

% β: %Beta cell activity; %S: %insulin sensitivity; AUC: area under the curve; HDL-C, high-density lipoprotein-cholesterol; HOMA: homeostasis modeal assessment; ISI: Insulin sensitivity index; LDL-C, low-density lipoprotein-cholesterol; TG, triglycerides; Fasting G:I, fasting glucose to insulin ratio.

fitness. All results were independent of significant changes in body composition.

Peak oxygen consumption and power output are considered reliable indexes of cardiorespiratory health and ACE is the most established and widely validated upper extremity exercise test.[30,46–48] Peak oxygen consumption is markedly reduced in SCI compared to the AB population.[22,49] Our study and previous research has shown ACE provides reliable changes in aerobic fitness in the SCI population.[25,36,50] El-Sayed *et al.* showed 12 weeks of ACE at 60–65% VO_{2peak} in chronic SCI improves aerobic fitness but did not control for completeness or level of injury.[32] Similarly, Sutbeyaz *et al.*[51] showed 6 weeks of ACE at 75% VO_{2peak} in chronic SCI improves aerobic fitness in subjects with T6–12 injuries. de Groot *et al.*[35] demonstrated 8 weeks of high intensity ACE showed more dramatic changes in VO_{2peak} than low intensity ACE, and DiCarlo[52] showed that 8 weeks of ACE in cervical SCI subjects improved VO_{2peak}. However, none of these studies controlled for completeness of injury. Despite an overall lack of homogeneity, systematic reviews agree that ACE improves aerobic fitness.[53–56]

It is well established in SCI literature that a level-dependent impairment of the respiratory system exists.

More cranial injuries display worse functional outcomes in numerous pulmonary measures.[57–59] This level dependent impairment is due to obstructive and restrictive lung disease caused by denervation of the pulmonary musculature, denervation of stabilizing abdominal musculature, and sympathetic blunting.[60] Motor-incomplete individuals (ISNCSCI C or D) have some pulmonary and skeletal muscle function below the LOI, while those individuals with a motor-complete injury (ISNCSCI A or B) do not. Individuals with incomplete injuries and/or lower LOI, may have disproportionate increases in cardiorespiratory function and therefore skew the outcomes of studies. The current study controls for completeness by only including motor complete injuries and controls for LOI by only including individuals of the same functional level, C7-T5, a level that is higher than what is needed to approach pulmonary function in the able bodied individual (T6–8).[57] The current study showed a 13% and 19% increase in resting VO_2 and relative VO_{2peak}, respectively, demonstrating the subjects increased ability to uptake, transport, and utilized oxygen after 10 weeks of training at 70% VO_{2peak}. It currently remains unclear whether the improvements in aerobic fitness stem from (1) physiological adaptions in heart rate, stroke volume, and/or cardiac output, (2) improvements in a-VO_2 differences (oxygen extraction in the peripheral tissues), or (3) neurological adaptions in the brain and/or cord. However, a combination of these factors is more likely.[61]

The physical strain of performing activities of daily living (ADLs) and community mobility is related to level of injury and physical capacity.[24,25,49,62,63] Individuals with a higher physical capacity (higher VO_{2peak}) are better able to perform ADLs, navigate the community, and have fewer medical complications.[24,25,49,62–64] The 12-minute wheelchair propulsion test is a reliable and cheap measure of community mobility.[42] DiCarlo showed 8 weeks of ACE improved 12 minute wheelchair propulsion from 1.18 to 2.1km on average in a cohort of eight subjects with cervical SCIs, but included congenital lesions and did not control for completeness.[52] The current study noted an average increase in propulsion of 336 feet, or 14%, a significant improvement that further validates the use of ACE to improve community mobility.

Sedentary individuals with SCI have poor metabolic profiles compared to the AB population,[2] while active individuals with SCI have more favorable profiles.[65,66] HOMA is a tool used to analyze fasting glucose and insulin levels. %β and %S are inversely related. HOMA-IR is the ratio of %β to %S. The average person in a population is represented by %β and %S of 100%, with an IR of 1.0. HOMA-IR highly correlates to insulin resistance measured by more invasive methods such as hyperinsulinemic-euglycemic clamp and intravenous glucose tolerance test.[67] The HOMA-IR cut-off value consistent with 66th percentile of insulin resistance in the AB population is HOMA-IR > 2.73.[68] HOMA measures are suggestive of fasting hepatic insulin sensitivity,[69–71] while Matsuda Index is a measure of whole body peripheral insulin sensitivity adaptations.[72] The current study found significant changes in post-intervention HOMA-%S (73.% to 105.6%) and HOMA-IR (1.6 to 1.0), which are consistent with previous research examining the effect of upper body exercise on hepatic insulin sensitivity.[38,39,73]

Approximately 70% of glucose intolerance post-SCI is due to intramuscular fat (IMF) accumulation and skeletal atrophy of the thighs.[74] One proposed mechanism to improve glucose tolerance is to decrease IMF by increasing LBM and decreasing FM.[75] Functional electrical stimulation (FES) of the lower extremities has the potential to alter cardiometabolic profiles from a multifaceted approach; skeletal muscle hypertrophy, non-insulin dependent glucose uptake, peripheral insulin sensitivity, and alteration of total and regional body composition. Previous studies have shown FES can increase regional and total body LBM as well as alter glucose and insulin metabolism, although they have not consistently shown a decrease in IMF.[36,76,77] ACE does not appear to activate enough muscle mass in the upper extremities to cause changes in body composition[53,78] but improvement in metabolic profile has been reported without changes in body composition,[33] and is supported by the findings of this study.

Extensive changes in FFM, such as those noted with FES-LCE, would be expected to cause a greater degree of change in metabolic profiles. The current study noted similar changes in metabolic profile to those with robust muscular hypertrophy of the thighs. FES-LCE does not reliably decrease FM,[36,76,79,80] which may result from the lack of dietary control in study design. When diet is controlled for, lower extremity FES has been shown to increase LBM, decrease IMF, improve fasting insulin, glucose AUC, insulin AUC, and lipid profiles.[81]

Study limitations

This study is not without limitations. (1) This study is of small sample size with only six participants completing the entire study raising the possibility of a type 2 error. However, this sample size is consistent with exercise-based SCI research.[36,37,43,81–84] (2) There is a lack of comparative control group. (3) ACE does not appear to cause body composition changes to the degree of

FES-LCE, resistance training, or FES-rowing.[33,36,81] However, FES equipment is more expensive than ACE, individuals have limited access to facilities with the equipment, transportation barriers, and environmental constraints limiting its use.[85] SCI individuals appear to adhere to FES and ACE when they have the opportunity to do it in their home.[40,85,86] (4) The diets of the enrolled participants were not controlled for. Lastly, (5) OGTT is not considered the gold standard in the assessment of carbohydrate metabolism, however the technique is less time consuming, less expensive, and is safer than the intravenous glucose tolerance test and hyperinsulinemic euglycemic clamp, especially for the SCI population.[87,88]

Conclusions

The findings suggest that ten weeks of 30minutes/day 3days/week ACE leads to an improvement in measures of cardiovascular fitness (resting VO_2, absolute VO_{2peak}, relative VO_{2peak}, RQ, peak power), community mobility (12-minute WC propulsion), and metabolic profiles (fasting insulin, G:I ratio, HOMA-S, and HOMA-IR) independent of changes in body composition. This work emphasizes the importance of exercise as a means to reduce CVD risk factors and improve aerobic fitness and community mobility.

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